Society Proceedings

PHILADELPHIA NEUROLOGICAL SOCIETY.

November 22, 1904.

The President, Dr. C. S. Potts, in the Chair.

A Case of Arteriosclerosis of the Nervous System.—This patient was exhibited by Dr. William C. Pickett. The man is sixty-seven years of age. Two years ago he began to be troubled with dizziness and headache; from that time he has slowly become "stiff all over," until now his walking is very difficult. Dr. Pickett had repeatedly questioned the family, and they insisted that at no time was there a sudden accession of these symptoms. The right leg and arm are decidedly more spastic than the left. Hemorrhage or thrombosis might be thought of, but the family insist that the symptoms have been gradual in onset. The knee-jerk is spastic and is greatest on the right side. Spasticity is much less in the arms and hands; but numbness of the fingers is complained of. The mental condition of the man is not impaired; his pupils react to light; bowels and bladder are well controlled; there are no sensory changes. Speech has become mumbling. It might be said that this is an ordinary case of senility, but Dr. Pickett thought we do not find such a history very often, and such cases have a new interest in the light of the somewhat modern conception of arteriosclerosis of the central nervous system. It seems to him that this case is not one of softening in the grosser sense, but must be one of arteriosclerosis, causing ischemia and impaired nutrition of areas, which, if thrombosis had occurred, would be the seat of softening, but in this slower process are the seat of degeneration and sclerosis. In the insane department of the Philadelphia Hospital, within a year, there have been two cases which might have been called simple demented paresis, but were different from any cases Dr. Pickett had ever seen. The history was that of a slowly increasing general spasticity with mental failure, and in one case, at autopsy, there was such a degree of sclerosis and atrophy of the frontal lobes—the prefrontal and anterior central gyri—that the contrast with the part of the brain back of them made a shelf-like projection. The explanation clinically and at autopsy was found in the arteriosclerotic brain-degeneration of Alzheimer. The arteriosclerosis of the larger vessels of the brain, the anterior cerebral, etc., was very striking.

Dr. Spiller thought that arteriosclerosis does produce great change in the brain in many cases, causing just such changes as in Dr. Pickett's case, and he said it is important to recognize them.

Dr. C. L. Allen thought that in most institutions where there are many old people, especially senile insane, in a certain proportion there will be symptoms incident to arteriosclerosis. He stated that he had recently had two cases of senile dementia, a man and a woman, presenting the picture of spastic paraplegia. The man died, and autopsy was denied, but he had arteriosclerosis, and the symptoms were probably due to arteriosclerotic changes in the spinal cord. The woman is still living, is bedridden, and has contractures of legs and arms. Before her symptoms became so marked she had increased reflexes and spasticity, which became greater and greater, until now the legs and arms are in a state of contracture. He regarded these cases as arteriosclerosis of the spinal cord.

He thought that many cases of vertigo and epilepsy could be explained by arteriosclerotic changes, and most of these cases, if they come to autopsy, show considerable arteriosclerosis.

Dr. Gordon thought that Dr. Pickett's case was probably one of arteriosclerosis. He cited two cases of his own in which he attributed the symptoms to arteriosclerosis. One of the cases had loss of reflexes, and the other loss of reflexes on one side and exaggeration on the opposite side, and still the arterial changes were uniformly distributed in both cases. He thought it hard to understand why, in some cases, there will be involvement of the posterior columns, and in others of the lateral tracts, while the arterial changes are equally distributed.

Hemiplegia Following Typhoid Fever.—This case was reported by Dr. C. S. Potts. F. L., aged twenty-eight years, family history good, previous history good. He had typhoid fever four years ago, and while convalescing became suddenly paralyzed on the right side. He was out of his mind three weeks immediately following. Speech was affected for ten weeks. When he came to the dispensary he had a right-sided hemiplegia, increased reflexes, Babinski's sign, ankle clonus, and right-sided paralysis of the face.

Dr. Potts thought the cause was either apoplexy or encephalitis, the three weeks of delirium being in favor of the latter.

Dr. Camp, in discussing Dr. Potts' case, exhibited a case from Dr. Spiller's service at the Polyclinic Hospital, which he thought might have sufficient connections with Dr. Potts' case to be of interest. The patient is twelve years of age. His family history is negative, and his previous medical history contains nothing of importance. He had scarlet fever at seven years of age, and had previous attacks of chorea. On May 4 of the present year he developed attacks of chorea. No blood examination was made, but the symptoms were typical. On May 19 he stopped using the right arm and leg, and could not lift them from the bed. At the same time they became the seat of irregular jerky movements. The doctor in attendance was not sure that the face was involved. On May 29 he developed a severe attack of pneumonia, which had its crisis on the 6th of June. He began to use the right arm normally as soon as he sat up in bed after the pneumonia. He was much emaciated and went to the country. On his return, seven weeks ago, he seemed perfectly healthy. Two weeks ago his mother noticed that he began to drop things from his right hand, and would not use the hand. He also became peevish and irritable.

When examined, November 16, 1904, it was noticed that the right side of the face was weaker than the left, although the upper part of each side of the face was normal. The tongue deviated to the right when protruded. Saliva dribbled from the left corner of his mouth. His knee-jerks were normal on each side. There was no Babinski reflex, although there was a tendency toward permanent hyperextension of the toes. Oppenheim's reflex was not obtained. Choreiform movements of the right arm and leg were distinct. He was given Fowler's solution, gtt. iii. tid. The choreiform movements seem now to involve also the face. The electrical reactions are normal. There is no atrophy. The diagnosis of his condition last May rests between hemiplegia developing during typhoid, or typhoid fever with a coincident attack of chorea. The condition at present may be a weakness remaining from the hemiplegia and now complicated by chorea, or it may be a case of paralytic chorea. There is also another possibility, there may be a lesion in one of the superior cerebellar peduncles, which, as is well known, will produce choreiform movements of the affected side.

Dr. Potts added that his patient had recovered the power of speech, and is now mentally bright. He said that L. Stein (*Pest. Med. Chir. Presse,* Jan. 10, 1904, p. 44) had recently reported a case of hemiplegia occurring during typhoid fever, and had commented upon the rarity of this sequel.

Dr. Gordon said that he had recorded, last year, a case of hemiplegia occurring during convalescence from typhoid fever. The symptoms came on suddenly, but the patient recovered entirely. These disturbances occurring in the last period of typhoid fever are only transient in character, according to the literature and his own observation.

Dr. Camp added that his case now presents the symptoms of a paralytic chorea, hemiplegic in distribution. It is rare to have the face involved in such cases. In the present case the face had been distinctly involved when first seen, which, if it is a case of paralytic chorea, marks it an exceeding rare one. On a careful review of the literature, published in the June number of the *University Medical Bulletin*, he was unable to find a case of paralytic chorea in which the face was involved.

A Case in which the First Left Temporal Convolution was Destroyed in an Adult Without Causing Word Deafness.—This case was reported by Dr. W. G. Spiller and Dr. H. B. Allyn. The patient, a man fifty-seven years of age, right-handed, had had two apoplectiform attacks, one in 1881 and another in 1886. Notwithstanding these attacks he could understand all that was said to him, could relate anecdotes and showed very few symptoms of aphasia in his later life. He was a man of considerable mental ability. At autopsy it was found that there was a complete destruction of the first left temporal convolution. The second was well developed. We must assume that the retention of function of word hearing was due to the preservation of the second left temporal convolution, or to the unusual development of the first right temporal convolution. A very similar case has recently been reported by C. S. Freund.

Dr. Mills thought an interesting point in connection with the specimen exhibited was that there appeared to be no arrest of development of the hinder part of the third frontal convolution, which was apparently of normal size. He thought some explanation might be reached indirectly from this fact if the man were a right-handed individual, as it appeared he was. He stated that in a case of his own, and in other cases reported of word deafness, after the lapse of years secondary involution of the third frontal convolution and other parts concerned in the mechanism of speech was present. He thought it looked as though this man's word-hearing center might be on the right side, rather than on the left, where it usually is in right-handed persons. He stated it as his belief that the lower portion of the hinder part of the first temporal convolution and the upper part of the second temporal convolution are concerned in word-hearing. He thought the explanation of this case lay in the unusual development of both temporal regions.

Dr. Dercum cited a case of Bramwell's in which there had been an attack of motor aphasia, but in which a short time subsequently the man spoke as well as before. At the autopsy it was found that there was a complete destruction of the third frontal convolution of the left side. Here was a case in which the right side of the brain had been functionally active as well as the left. He thought it possible that in persons unusually gifted there may be an education of both centers; that is, such persons would have ambidextrous or, more properly speaking, ambisinistral brains.

Dr. Spiller stated that the view brought forward by Dr. Dercum was Freund's explanation of his case, that the word-hearing center had been latent on the right side, and was called forth when unusual demands were made upon the brain. Referring again to the case reported by Dr. Allyn and himself, Dr. Spiller said that he had asked Dr. Allyn particularly about the man's appreciation of music, because so little is known about the center concerned in the hearing of music. The patient had not been musical. The anterior part of the first left temporal convolution is supposed by some to be the seat of music hearing, while the center for word hearing is in the posterior part of this same convolution.

A Case of Multiple Spinal Sclerosis, with Remarks Upon the Pathogenesis of the Disease.—This paper was read by Dr. F. X. Dercum and Dr. Alfred Gordon.

The Earliest Cases of Disseminated Sclerosis with Necropsy and Microscopical Examination Reported in America.—Dr. Charles K. Mills thought it would be well, from a historical standpoint, to refer to the fact that the first cases of multiple sclerosis with necropsy were recorded by E. C. Seguin, of New York, in 1878; and that he (Dr. Mills) in 1879 reported a case with necropsy and microscopical findings in the *Hospital Gazette* of New York. As the cases both of Dr. Seguin and himself were apparently not well known to American neurologists, it might be well to call attention to them in some detail.

The notes of Dr. Seguin were on two cases, which were recorded in a paper which was entitled: A Contribution to the Pathological Anatomy of Disseminated Cerebrospinal Sclerosis, this having been presented at a meeting of the New York Neurological Society, held February, 1878. The paper was published in the JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. 5, April, 1878.

The first case was that of a man twenty-nine years old. His first symptoms were disturbance of vision and incoordination after marching. In less than a year his symptoms were well marked. The case is well recorded, although not as regards studies in sensation, reflexes, coordination, etc., exactly after the manner in which a record of to-day would be made. Examination of the reported facts, however, show that the patient had a spastic, paretic gait, incoordination both in the lower and upper extremities, intention or action tremor, nystagmus, irregular disturbance of the sensation, exaggerated reflexes, disorder of speech, and other symptoms clearly pointing to disseminated sclerosis. The man died a little more than six years after the first symptoms were observed.

The second case was that of a young woman, twenty-three years old. She first began to suffer with weakness of the right leg, which developed into a marked paresis, sensibility to pain being a little dulled in the leg and foot. Dr. Seguin first believed that this case was a hysterical disorder, a significant fact when it is remembered how often hysteria and disseminated sclerosis have been confused. About six months after her first symptoms she somewhat rapidly became paraplegic, her hands also showing some paresis. Later bed-sores developed, apparently, according to the reporter, from want of care while under treatment in a hydrotherapeutic establishment. The autopsy was made by Dr. Seguin, the spinal cord only being removed. This second case was not nearly so well recorded clinically as the first.

The brain and spinal cord in Case 1 were hardened in potassium bichromate; sclerosis in patches was found in various parts of the cerebrum, caudatum and in the oblongata. The exact position and extent of the foci of sclerosis are given in numerous instances. Sections of the spinal cord were made from the cervical, dorsal and lumbar regions, these being stained with carmine and hematosin. The foci of sclerosis in various parts of the cord are described.

In the second case the microscopical examination was only of the cord, and Seguin gives a description sufficiently full to show that this, like the first case, was clearly one of disseminated sclerosis. In summarizing the cases he remarks that the sclerosis nodules were of various sizes, and occupied the most diverse regions of the cerebrospinal system, involving the gray as well as the white matter. He makes a differential study of the histological appearance, as observed in an early stage, in one in which the morbid process has considerably advanced, and in a third in which the most extensive changes are to be seen. Descriptions are given of the appearance presented by the neuroglia, nerve cells, vessels, etc., and a plate, including sixteen illustrations, is given. The histological descriptions are good for the time when the investigation was made.

It will be remembered that Seguin had pursued his clinicopathological studies in the wards and laboratory of Charcot, and also later with Forel. Certainly the record of these cases should not be overlooked in speaking of the cases of disseminated sclerosis recorded in this country, even though it is true that the more recent work of Spiller and others regarding the same disease has been of a more exact and elaborate kind.

The case reported by Dr. Mills in 1879 was one which had been carefully studied clinically and one in which a necropsy had been obtained and a subsequent microscopical investigation made. The case proved to be one of disseminated sclerosis with diffuse, and in some places coalescing patches in the dorsal and lateral columns. This case had not attracted attention in the résumé of the literature of disseminated sclerosis and combined sclerosis, probably in the main because of the place of its publication. It was published in consecutive numbers of the *Hospital Gazette of New York,* Nov. 22 and Nov. 29, 1879. The journal was chiefly devoted to the publication of clinical lectures and other clinical material, and while its circulation was large, it had only a brief existence, and probably is seldom consulted by neurologists and writers on neurological topics.

The patient was a woman, thirty years old, whose past history was uncertain, but who said that the first symptoms of her disease came on about a year before her admission to the hospital after an attack of typhoid fever. It seemed probable that the invasion of her disorder was earlier than the time fixed by the patient. The dominating features of the case were spasmodic paresis and ataxia, both lower and upper extremities being highly spastic; knee-jerks and other deep reflexes were exaggerated; intention tremor was shown when the patient attempted to walk; it was also shown in her efforts to use her upper extremities. Her speech was distinctly of the scanning and syllabic character; she showed some mental hebetude and deficiency; partial atrophy of both optic nerves was present but nystagmus was absent. The changing emotional states which accompany disseminated disease of the pons and oblongata were particularly marked. Impairment of sensibility, especially in the lower extremities, was determined by careful investigation. When most of the examinations were made the ataxic symptoms were largely masked by the spastic manifestations. During the two months preceding her death she grew rapidly worse. The spasticity markedly increased, she became more and more helpless, and was soon totally bed-ridden. The complaints of pain, especially in and about the knees and elbows, became more frequent, and now and then she would have terrific stabs of pain in her legs, thighs, arms or trunk. Her speech and mental strength failed steadily, involuntary evacuations began two weeks before her death. In the last five days of her life her knees and ankles were red, hot and swollen, like those of a person suffering from acute inflammatory rheumatism. A sacral bedsore formed, and other similar sores at the ankles and on the feet. Both brain and cord were examined carefully in the fresh state and after hardening. Besides the appearances of a chronic disseminated sclerosis, the microscope revealed evidences of a recent acute or subacute inflammatory affection of both the cord and brain. Sclerotic nodules were described as occurring in both thalami, in the crura, pons and oblongata. The lumbar enlargement and lower portion of the thoracic cord presented a shrunken and atrophied appearance even to the naked eye. The results of the microscopical examination of the cord, oblongata, basal ganglia and cerebral hemispheres are given, these showing typical advanced disseminated sclerosis. The extent and position of the sclerotic areas are described in detail.

Dr. Camp referred to two cases of multiple sclerosis with autopsy, reported by Dr. Spiller and himself. He stated that in these cases the incontinence of urine and feces was inconstant, while in Dr. Dercum and Dr. Gordon's case they were persistent, and that this was probably due to the more advanced stage of the disease. In both of the cases Dr. Spiller and he had reported symmetry of the pathological lesions was a marked feature, which was different from the findings mentioned in Dr. Dercum and Dr. Gordon's case. As regards the theory of the vascular origin of the disease Dr. Spiller and he had found the blood vessels in the sclerotic areas much thickened, but outside these areas they were practically normal, which would point to at least some connection between the two.

Dr. Dercum thought the facts were not such as to justify the belief that this disease is due to developmental defect. On the other hand he did not think it had its origin in the nervous elements or blood vessels. We are driven to the conclusion that the disease begins in and remains most pronounced in the glia. Its origin is still a matter for speculation.

Dr. Spiller said that in one of the early cases under his care there was secondary deegneration and ophthalmoplegia. During life the diagnosis of multiple sclerosis was made. He called attention to another view held in regard to the nature of this disease, namely, congenital malformation of the spinal cord and brain. Exposure to cold and damp is also important. This had been borne out in his own experience, as there was a history of exposure to either cold or dampness in every case he had had. The case reported by Drs. Dercum and Gordon had entered the hospital under Dr. Spiller's care, and the diagnosis of multiple sclerosis had then been made. Later the case had been in the service of Dr. Potts.

Dr. Gordon stated that he was familiar with the influence of damp and cold upon this disease. He stated that there was a suspicion of specific disease in the case they had reported. The woman had never had living children, had several miscarriages, and cystic ovaries. There was also a history of trauma. They, therefore, had no solid basis as to formation of an opinion upon the etiology of multiple sclerosis, as multiple factors enter into the etiology of the affection.

A Case of Tic Convulsif (?) in a girl of Six, and a Case of Hemiparesis, Muscular Rigidity and Exophthalmus with Von Graefe's Sign.—These cases were reported by Dr. Joseph Sailer.

Dr. Gordon said that although Dr. Sailer had stated there was no hysterical stigmata, he did not think that excluded the possibility of the case being one of hysteria. The typical symptoms of Gilles de la Tourette's disease were wanting, particularly echolalia and coprolalia. The complete disappearance of the symptoms under the influence of a small dose of a drug, speaks very much in favor of hysteria.

Referring to Dr. Sailer's second case, with regard to the presence of Von Graefe's sign in other conditions than exophthalmic goiter, Dr. Camp stated that he examined a case in Dr. Spiller's clinic in the Philadelphia Hospital, this fall, in which Von Graefe's sign was very distinct, and which was not a case of exophthalmic goiter.

Dr. Sailer called attention to the fact that he had been careful to put a question mark in the title of his first case, and that from a further study of the case he was inclined to believe it to be one of hysteria, but he thought even that diagnosis did not seem entirely satisfactory.

Specimens from a Case of Fracture of the Fifth Cervical Vertebra, Causing Total Transverse Lesion of the Cord.—These were exhibited by Dr. F. X. Dercum.

T. W., male; black; aged twenty-four; laborer; was admitted to the Nervous Wards of the Philadelphia Hospital, November 14, 1904. Clinical diagnosis, fracture of the fifth, sixth and seventh vertebræ, with hematomyelia of the same region. Chief complaint, paralysis of trunk and limbs, caused by an iron bucket falling on the head and neck. The family history is unimportant and negative, as is also the previous medical history. The patient was never ill in his life before. Has never had syphilis; thinks he had gonorrhea once when very young. Has used alcohol and tobacco very moderately.

History of Present Illness.—About 4 P.M. of July 13, 1904, the patient was engaged in pulling up buckets of cement to a height of seven stories, each bucket weighing from seventy-five to one hundred pounds. He was struck on the back of the head and neck by the bottom of a bucket, which had descended suddenly and very rapidly. He immediately became paralyzed over his entire body. Control of the bowels and bladder was lost at the same time. He was taken to the Medico-Chirurgical Hospital, and remained there until his transfer to the Philadelphia General Hospital. He was, according to his own statement, entirely conscious at the time of the accident. Shortly after, however, he became delirious, and remained so about three days. At the Medico-Chirurgical Hospital a loss of sensation was noted, involving the trunk from the nipples down anteriorly and posteriorly from the lower border of the scapulæ. This loss was total and included all forms of sensation. Over the arms there was an absence of sensation extending from the elbows to the ends of the fingers. There was also incontinence of urine and feces. An X-ray examination revealed a fracture involving the fifth, sixth and seventh cervical vertebræ. There was no abnormality of any of the viscera. Patient was able to take food and to digest it well.

Notwithstanding the fact that he was placed upon a water-bed, a bedsore developed over the buttocks on July 27. By August 15, similar sores made their appearance over the heels and ankles, by September 1 over the heads of the fibulæ and elbows, and by October 10, two small bedsores over the left scapulæ appeared.

At the time of his admission to the Philadelphia General Hospital the patient complained of a sensation of cold, involving the entire body and all his extremities. The penis was in a state of priapism. Bedsores noted at the Medico-Chirurgical Hospital were also present on admission, and in addition were sores in both popliteal spaces and another over the right shoulder.

Physical Examination.—The patient was a well-nourished negro, of twenty-four. Mucous membranes were of good color. The skin over the malar bone had taken on a whitish color. Over the entire body, but more especially on the feet, legs and upper extremities, the skin was much thickened, hard, and showed evidence of desquamation. The head was of normal shape, and covered with an abundant supply of curly black hair. There was a depression of the skull just over the occipital prominence on the left side; this depression was not deep, but easily made out; the face did not show any asymmetry nor palsy; the patient was able to show his teeth, draw the corner of his mouth to each side, and the naso-labial folds were of equal prominence. The patient could talk; answered questions well; whistled and wrinkled his forehead. He had all of his teeth, which were in good condition. Deglutition was normal. The patient laid flat on his back, with his upper extremities lying across his chest, in a state of contracture. He was unable to move any part of his body, and if an attempt were made to raise any limb, or disturb him in any way, he seemed to suffer pain. His chest was well developed; respiratory excursion was good on each side. The lungs were resonant throughout; there were no adventitious sounds. Respiration was diaphragmatic; pulse was rapid, full and regular. The heart was normal in outline; the apex was at the fifth interspace in the mid-clavicular line; the mitral sound was normal; there were no murmurs. There was a slight accentuation of the second aortic sound.

Eyes.—The pupils were equal, reacted to light, accommodation and convergence rather promptly. No extra-ocular palsies were noted. The conjunctivæ were discolored yellow. The tongue was protruded promptly in median line; was clean, and slightly fissured.

Back.—On examining his spinal column, patient had to be turned very gently on his right side, and in going over spinal column, from below upwards, tenderness to pressure existed over the first dorsal vertebra, and thence upward over the cervical vertebræ, until the fourth was reached. This seemed by all means the most painful. No crepitus could be elicited. The patient was unable to turn his head; his neck seemed to be rigid.

Arms.—When an attempt was made to raise the arms, pain was experienced by the patient, and when they were raised they fell heavily down upon the chest. The arms were in a state of contracture. The patient was unable to perform any movement with the arms; the grip in each hand was gone. The skin over the hands was thick, hardened and desquamating. The reflexes were abolished. There seemed to be some atrophy. Sensation in upper extremities was lost up to the junction of the middle and upper thirds of the arm.

Legs.—The patient was unable to raise either limb from the bed, and when an attempt was made the disturbance gave pain. The patient was unable to perform any movement with the legs. The reflexes on each side were abolished. Irritation of the plantar surfaces did not give any response. The skin over the feet and toes was much thickened, hardened and desquamating, as over the hands. Each foot was markedly swollen, and somewhat edematous. Sensation in each extremity was lost. The penis was in a state of semi-priapism; the glans was swollen and excoriated.

Sensation in all forms was lost over the entire body anteriorly as far upward as a line drawn from shoulder to shoulder, and posteriorly by a line drawn from the upper border of one scapula to the other. Above this level, anteriorly, sensation was normal, but posteriorly there was marked hyperalgesia over the entire cervical region. Muscles of the trunk were paralyzed. Both sphincters were paralyzed.

Trophic Changes.—The entire body had a cold feeling, the skin was thickened and hardened over extremities as has been described. The nails were brittle and ridged. Bedsores were present as described. Unilateral sweating of forehead on right side was also noted.

November 7, 1904.—For last three or four weeks the patient had been complaining of sharp pains radiating from right ear down to lower part of the mouth, but only during mastication or yawning. The patient also stated that there was a "watering" from both eyes, but especially from the right eye. He complained of pain in upper part of the spine, between the shoulders, and radiating to both arms.

November 9, 1904.—To-day there was free perspiration on left side of body, covering face, neck, arms, forearms, hands and trunk.

November 10, 1904.—The patient was very restless and noisy during the night; he twitched over the entire body. He complained of difficult swallowing, and his respirations were labored. He also complained of pain of a severe character in his neck, and at times over his entire body.

November 10, 1904.—Eye examination by Dr. de Schweinitz. No change in optic discs, both of which are pysiologically cupped. The veins and arteries are about normal in size; the arteries are exceedingly pallid, the veins dark. Roughly tested the visual fields are normal in both eyes. The pupillary reactions are normal. No paralysis of any external ocular muscles.

November 12, 1904.—Patient had been expectorating a good deal of mucus. Examination of the lungs showed moist râles over the bases of the lungs. The temperature of patient had been rather sub-normal since yesterday.

November 14, 1904.—Patient died at 6.45 A.M.

Autopsy Records.—Pathological diagnosis, fracture of fifth cervical vertebræ. Acute cystitis; edema of lungs; cloudy swelling of kidneys.

Body of an adult, male, negro. Extremities considerably emaciated. Over both elbows, posteriorly are two ulcerated areas, about three inches long, by two wide, both communicate with the elbow joint, the surfaces of which are much roughened. Over both scapulæ are small areas of ulceration. Over the buttocks there is a bedsore about eight inches in diameter. On the outer surfaces of the tibia are ulcerrations and likewise over the external malleoli.

The peritoneum is smooth and glistening. There is a slight amount of clear yellow fluid. The appendix extends downward, and is free from adhesions.

The lungs are distended, and nearly meet in mid-line. Both pleuræ contain a slight amount of clear yellow fluid. There are a few easily broken adhesions at both apices. The left lung is pale in color, soft, crepitant, from cut surface pale frothy fluid escaped on pressure. The right lung, lower, middle and posterior portions of upper lobes, purplish in color, firm, non-crepitant; from cut surface exuded a large amount of slightly tinged fluid, free from air. A bronchial gland showed caseous degeneration.

The pericardium is smooth and glistening and contains about 50 c.c. of clear fluid. The heart muscle is very pale in color, but firm. Some fibrous change is noted at the tips of the papillary muscles. The endocardium is normal.

The spleen is enlarged, pale in color; capsule smooth and glistening; cut surface shows trabeculæ and bodies quite prominent.

Kidneys considerably enlarged, pale in color; capsule strips easily; cortex thickened, numerous red striæ present; pyramids much injected.

Bladder.—Mucous membrane dark in color, and shows numerous irregular areas of hemorrhage; in one or two places the mucous membrane is absent.

Liver very pale in color, cut surface shows some congestion of vessels. Common bile duct patulous.

Small intestine throughout its entire extent shows marked congestion. Large intestine shows a few areas of congestion.

The brain shows no other gross changes than marked anemia.

The spinal column shows a transverse fracture of the fifth cervical vertebra. Fragments displaced. Cord firmly compressed at this point.

It was probably the sixth segment of the cord that had been destroyed, though the lesion had probably invaded also the fifth. The fourth cervical segment had evidently escaped, thus permitting diaphragmatic respiration to continue so many months. The level of the anesthesia is about that which one would expect to find in injury of the sixth and fifth cervical segments. The level of the anesthesia noted at the Medico-Chirurgical Hospital, immediately after the accident, was lower than that subsequently noted by Dr. Dercum. Doubtless this is to be explained by extension of inflammatory and degenerative changes upward above the site of the original lesion.